Proposed Specifications for a Lumbar Spinal Cord Electrode Array for Control of Lower Extremities in Paraplegia

Vivian K. Mushahwar, *Member, IEEE,* and Kenneth W. Horch, *Member, IEEE*

Abstract—**The goal of the study was to provide specifications for a stimulating electrode array to be implanted in the lumbo-sacral spinal cord as part of a functional neuromuscular stimulation (FNS) system for control of lower extremity muscles in paralyzed individuals. Dual channel stimulation of the quadriceps activation pool in the feline ventral lumbo-sacral spinal cord was performed to measure electrode interactions and to explore the effect of various stimulation paradigms on muscle fatigue. There was no measurable overlap in the populations of motor neurons activated from two different electrodes for spacings $\geq$ 1 mm with currents below 100 μA. However, a statistically significant increase in the population of activated fibers due to current summation was observed when stimuli $\geq$ 70 μA were simultaneously presented through pairs of electrodes within 3 mm of each other. Fatigue effects were studied with three paradigms: 1) stimuli were delivered through a single electrode, 2) stimuli were delivered through two electrodes with the stimulus to the second electrode presented during the refractory period of fibers stimulated by the first electrode, and 3) stimuli were interleaved between the two electrodes such that the stimulus to one electrode was presented midway between stimuli to the other electrode, and the rate of stimulation through a single electrode was half that used in the first two paradigms. Dual channel refractory and single channel stimulation did not differ from each other in the rate at which the muscle fatigued, in both cases the force decayed to 30% of its initial level within 2 min of the initiation of the stimulation regime, whereas the force with interleaved stimulation was still above the initial force at this time due to strong potentiation. Based on these results and on and activation pool dimensions obtained in an earlier study, preliminary specifications are presented for an electrode array to be implanted in the human spinal cord for functional neuromuscular stimulation.**

Index Terms—**Functional electrical stimulation, motor neurons, spinal cord stimulation.**

I. Introduction

SPINAL cord injuries cause an interruption in the transmission of neural signals from the brain to the periphery leading to the loss of voluntary control of muscles innervated by motor neurons originating below the level of the spinal cord lesion. Artificial control of paralyzed muscles has been partially accomplished using functional neuromuscular stimulation (FNS), whereby neuromuscular junctions or peripheral nerves are electrically stimulated using motor point or cuff electrodes. Though some success has been achieved by current FNS systems, several drawbacks remain. These include frequent lead breakage [1], dependence of muscle response on location of electrode implantation relative to the motor point [2], and muscle fatigue with the consequent inability to sustain fused contractions for adequate periods of time [1], [3]–[5].

We have suggested [6], [7] the use of spinal cord stimulation in FNS for the following reasons. The spinal cord is distant from contracting muscles, so electrodes implanted therein will not be subjected to damaging stresses and strains due to movement of the target tissue. The lumbo-sacral spinal cord is compressed allowing activation of essentially all lower extremity muscles by implanting electrodes in a relatively small and protected region. Selective activation of muscle groups can be obtained by stimulating their "activation pools" (regions in which focal electrical stimulation elicits contraction in specific muscle groups in isolation) in the ventral horn of the spinal cord. Smooth and graded contraction of muscles, with near to physiological recruitment order of motor units, can be accomplished by focal stimulation of activation pools.

An efficient spinal cord stimulation system would consist of electrodes spaced far enough from each other that overlapping stimulation of motor units is minimized, yet close enough to allow for summation of subthreshold currents from the two electrodes to maximize motor unit recruitment and force output [3], [8]. Such an array would also allow different regions of a given activation pool to be accessed to allow rotation of mechanical work among different motor units to minimize fatigue for postural and other prolonged activities.

In the present study, we looked at stimulus interactions between pairs of electrodes in the feline quadriceps activation pool as a function of stimulus strength, timing and inter-electrode spacing. Using these data, and data on the dimensions and location of the activation pools obtained in a related study [7], [9], we have derived preliminary specifications for a human lumbar spinal cord electrode array to be used in FNS applications.

II. Methods

A. Experimental Setup

Five adult cats were used in this study. Anesthesia was induced with a 40-mg/kg intraperitoneal injection of sodium pentobarbital and was maintained with intravenous injections

Manuscript received September 27, 1996; revised May 4, 1997.

The authors are with the Neuroprosthetics Laboratory, Department of Bioengineering, University of Utah, Salt Lake City, UT 84112 USA.

Publisher Item Identifier S 1063-6528(97)06347-7.

of 1:10 dilution of the anesthetic as needed. A tracheotomy was performed on one cat, and a tracheal tube was inserted and firmly tied to the trachea in preparation for decerebration later in the experiment. Each cat was placed on a heated plate and its body temperature was monitored through a rectal probe and maintained near 37 °C. The cat's left leg and back were shaved and the animal was positioned in a Kopf spinal unit. The spinal cord was exposed from lumbar segment L4 to L7. The dura mater was removed with iridectomy scissors and the spinal cord was covered with saline to prevent its dehydration.

Steinman pins were placed in the cat's lateral femoral epicondyle and the medial proximal shaft and medial malleolus of the tibia and were clamped through posts to the heated plate. The patellar tendon was dissected from its point of insertion and attached to a force transducer (model SM-25, Interface, Scottsdale, AZ) that allowed isometric measurement of forces generated by the quadriceps muscle. To monitor activity in the remaining main hindlimb muscles, bipolar electromyogram (EMG) electrodes were implanted in the cat's biceps femoris and semimembranosus/semitendonosus muscles and a force transducer (model MB-5, Interface, Scottsdale, AZ) was placed on the foot pad to facilitate detection of movements around the ankle and paw. Only results from stimulation that did not activate neurons from other motor pools, as was evidenced by the absence of detectable EMG's in neighboring muscles or ankle torques, have been included here.

Comb stimulating electrode arrays were constructed from six tungsten rods, each 100 μm in diameter, sharpened, and insulated except for the tip. The rods were glued to one side of a microscope slide such that they all extended to the same depth and were spaced 1 mm apart. The electrode array, mounted in a micromanipulator, was advanced into the quadriceps activation pool in the ventral horn of spinal segments L5 and anterior L6 [7], [9]. An 18-gauge hypodermic needle placed in the right latissimus dorsi muscle served as the return electrode. Stimulus generation and response recording were performed by an 80486-based computer with appropriate analog-to-digital and digital-to-analog interfaces.

Prior to initiating the overlap and fatigue stimulation protocols, amplitude modulated, 600 μs long, biphasic stimuli with a 500-μs interphase interval were delivered through each electrode separately to determine the threshold current required to elicit the smallest detectable activity in quadriceps, and the maximum current ($\leq$ 100 μA) before stimulus spread to neighboring activation pools was noted. A "standard stimulus," which produced an intermediate level of muscle force, was delivered through the electrodes individually and the resulting quadriceps force (standard response) was recorded. Throughout the electrode interaction protocols, the fatigue state of the muscle was monitored by delivering the standard stimulus to the various stimulating electrodes and comparing the generated force to the standard response. If the generated force was not within 10% of the standard response, the muscle was considered fatigued and was given enough time to recover before resuming the experiment.

At the end of the experiments, electrolytic lesions were placed in the spinal cord to mark the stimulation sites. The cats were perfused through the heart with Palay's fixative and the lumbar spinal cord was removed. The cord was subsequently sectioned to verify that the electrode array was in the quadriceps activation pool.

B. Electrode Interactions

Electrode interactions were determined as a function of pulse amplitude and electrode spacing. In all of the cats, single 600 μs pulses were used to elicit twitch contractions with three different pulse amplitudes: 0.25, 0.50, and 0.75 of the way between the threshold and maximum currents for that stimulation site. In one cat, 760-ms long, 50 Hz trains of 300 μs duration biphasic pulses were also used to elicit tetanic contractions at the highest current level (100 μA) considered safe for intraspinal stimulation [10]. Four electrode spacings (1–4 mm) were used in the former cases, but only the 1-mm spacing was studied in the latter.

Three modes of stimulation were used: 1) stimuli were presented through each electrode separately, 2) stimuli were presented through two electrodes simultaneously, and 3) stimuli were presented through two electrodes with the stimulus to the second electrode delivered during the refractory period of fibers activated from the first electrode. Within a stimulus sequence, pulse amplitude and stimulation mode were chosen pseudorandomly (randomly without repetition) to eliminate the dependence of force generation on the order of stimulus delivery. An interval of 60 s was allowed between stimulus presentations.

C. Fatigue

The time course and magnitude of fatigue were measured for different stimulation patterns. The quadriceps was fatigued using the intermittent fatigue protocol described by Burke *et al.* [11]. An electrode spacing was chosen, and biphasic, rectangular, charge balanced pulses 300 μs in duration with a 500-μs interphase interval, were delivered in 760-ms long trains under three different paradigms: 1) a 50-Hz train was delivered to only one electrode at a time (single), 2) 50 Hz trains were delivered to both electrodes with the second electrode activated 400 μs after the end of stimulus to the first electrode (refractory), and 3) 25 Hz trains were delivered to each electrode in an interleaved manner such that the aggregate stimulation frequency was 50 Hz (interleaved) [12]. Simultaneous stimulation through two electrodes, each at 50 Hz, was not attempted in the fatigue study since, though lower stimulus amplitudes could be expected, the paradigm would lead to the activation of the same population of motor neurons around each electrode along with additional fibers due to current summation [12]. Such a paradigm is indistinguishable from the single electrode stimulation paradigm used in the study. In each case, pulse amplitudes were chosen to produce an initial-fused tetanic peak force around 15 N, a value that is typical of that seen when using stimulus intensities one-fourth of the way between threshold and maximal. The paradigms were selected in pseudo-random order within an experiment to eliminate the effect of order on the force output. In all cases, stimulation lasted for 5 min with a 1-s interval between

the end of one train and the beginning of the next, producing a total of 170 tetanic contractions.

As in the electrode interaction experiments, a "tetanic standard response," produced by stimulation through each electrode separately at a low pulse amplitude prior to initiation of the fatigue protocol, was used to assess the state of the muscle following each fatigue session. In general, a period from 60 to 120 min was needed for muscle recovery to within 10% of the standard response prior to resumption of the experiment.

D. Decerebration

To control for the possibility that the results were affected by the Nembutal anesthetic, one cat was decerebrated and removed from anesthetic support once the interaction and fatigue protocols were completed. At this point, the location of the electrode array was noted on the micromanipulator's scale and the array was withdrawn from the spinal cord. An opening was made in both parietal bones above the lamboidal ridge separating the cortex from the cerebellum while the cat was still under Nembutal anesthesia. A Pasteur pipette, connected to a vacuum pump, was used for aspirating the caudal portion of the cortex to allow for better visualization of the corpora quadrigemina. A blunt spatula was repeatedly inserted through each of the parietal openings to produce a precollicular lesion. A period of about two hours was given for the cat to return to a mildly reflexic state before the decerebration was completed. Once the decerebration was complete, the cat was artificially respired with its endtidal pCO_2 maintained at 3 to 4% and the electrode array was reinserted in its former location. For testing motor neuron excitability after the decerebration, three to six quadriceps tetanic contractions were produced by delivering 760-ms duration, 50 Hz trains of 100 μA, 300 μs long biphasic pulses with an interphase interval of 500 μs.

E. Data Analysis

Overlap in the populations of neurons activated from individual electrodes was calculated by

$$\text{Overlap} = \frac{F_a + F_b - F_{\text{ref}}}{F_{\text{ref}}}$$

where F_a and F_b are forces from each electrode separately, and F_{ref} is the force generated using refractory mode stimulation. Recruitment of additional motor neurons by summation of subthreshold currents during paired stimulation was quantified as

$$\text{Summation} = \frac{F_{\text{simul}}}{F_a + F_b}$$

where F_{simul} is the force generated when the stimuli were presented through the two electrodes simultaneously. Note that F_{simul} is the force obtained by activating the same neuronal populations involved in generating F_{ref} plus the force generated as a result of current summation in the subthreshold neuronal fringes due to simultaneous stimulation through two electrodes [3], [12].

The fatigue data were analyzed by combining the average plateau forces from five successive tetanic contractions into a single bin, giving a total of 34 data points for each fatigue curve. Each point was normalized relative to the force of the first bin. Data from the five cats was then combined and standard errors were calculated for the combined data.

F. Activation Pool Dimensions

In a related study [7], [9], the ventral lumbo-sacral spinal cord was mapped and the boundaries of the quadriceps, tibialis anterior and triceps surae activation pools were determined. For each cross section in a given pool, the distance from the spinal cord midline to the center of the pool, the distance from the spinal cord dorsal surface to the center of the pool, and the pool length, width, height and cross-sectional area were measured. The means of the distances to the center of the pool from the spinal cord midline and dorsal surface, and the width, height and length values obtained from quadriceps activation pools in six animals, and tibialis anterior and triceps surae pools in five animals each, were calculated and scaled to reflect the average size of the spinal cords in the cats used in the study. Because fine mapping of the hamstring activation pool has not been done, its location and dimensions were derived from coarse maps obtained in earlier studies [6], [7] in conjunction with the location and dimensions of the hamstrings anatomical motor pool reported by Romanes [13], [14].

III. Results

A. Electrode Interactions

There was no statistically significant overlap of stimulated neuronal populations for pulse amplitudes of 30, 50, and 70 μA and electrode spacings between 1 and 4 mm in data from the five animals (calculated overlaps ranged from 1.9 ± 14.5 to $8.7 \pm 23.9\%$, mean $\pm$ s.e.). On the other hand, a 100-μa stimulus produced an overlap of $15.4 \pm 2.3\%$ between electrodes 1 mm apart in the one animal tested at this level.

For currents below 70 μA (i.e., 30 and 50 μA), there was no measurable increase in muscle force due to summation of subthreshold currents when stimuli were delivered to pairs of electrodes simultaneously at any of the three separations (1, 2, and 3 mm) tested. However, there was statistically significant ($p < 0.05$) summation at 70 μA at all three separations, as shown in Fig. 1. As expected, this effect showed a significant ($p < 0.05$) dependence on distance. Summation was also seen with 100 μA stimuli.

B. Fatigue

To produce an initial tetanic quadriceps force of 15 N, the single, refractory and interleave fatigue stimulation paradigms required a mean pulse amplitude per electrode of 31, 34 and 46 μA, respectively. While the pulse amplitudes utilized for the single and refractory stimulation mode were similar, each was significantly lower ($p < 0.01$) than the amplitude used for interleaved stimulation. Fig. 2 shows pooled fatigue data from the five cats obtained with single, refractory and interleave stimulation paradigms. Electrode spacing varied

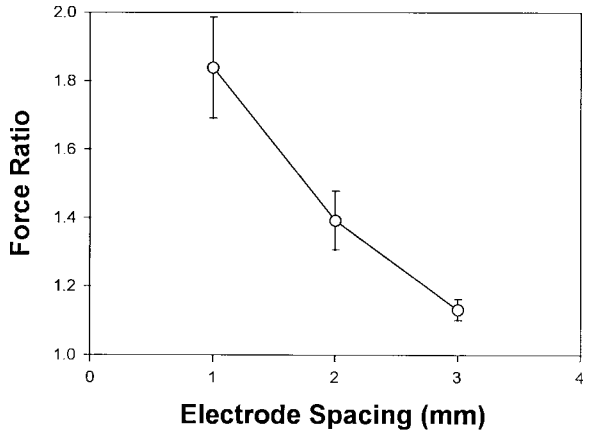

Fig. 1. *Recruitment by current summation.* Shown are the mean and standard error values of force increase during simultaneous stimulation through two electrodes compared to the sum of the forces generated by stimulation through each electrode alone as a function of electrode separation. Stimulus amplitude was near 70 μA in each of the five animals.

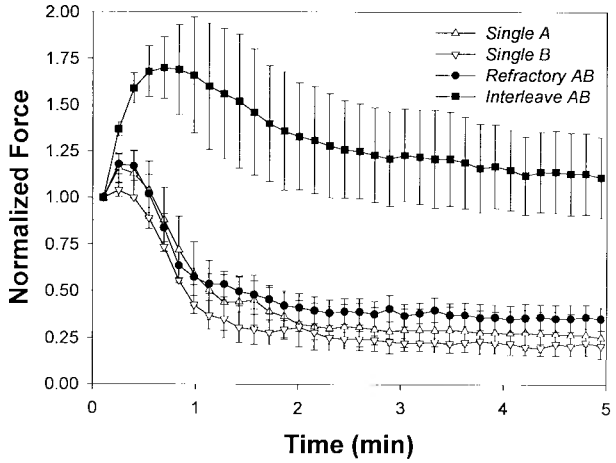

Fig. 2. *Effect of stimulation paradigm on muscle fatigue.* Shown are normalized tetanic forces produced by repeated pulse trains lasting 760 ms as a function of time. Each point shows tetanic contraction forces from five cats, binned for five successive stimulus presentations, and normalized to the value of the first bin. Four different stimulus paradigms were used. In *Single A* and *Single B*, 50 Hz trains were presented to a single electrode. In *Refractory*, 50 Hz trains were presented through both electrodes such that the stimulus to electrode B was delivered during the refractory period of neurons activated by electrode A. For *Interleave*, 25 Hz trains to the two electrodes were interleaved such that each electrode was stimulated in the middle of the period between stimuli to the other electrode.

from 2 to 4 mm between cats but was held constant within an animal. The initial increase in force during the stimulation sequences was due to post-tetanic potentiation [11], [15]–[18]. After the potentiation, single (*Single A* and *Single B*) and refractory (*Refractory*) mode stimulation produced muscle fatigue profiles that did not differ from each other statistically, but that were significantly ($p < 0.0001$) different from that produced by interleaved stimulation (*Interleave*). Within 1 min of intermittent stimulation, the force generated by single and refractory mode stimulation had fallen to half its initial value, and by 2 min it was further reduced to only 30%. In contrast, force generated by interleaved stimulation was actually elevated at 1 min, and was still above what it was initially even at 5 min.

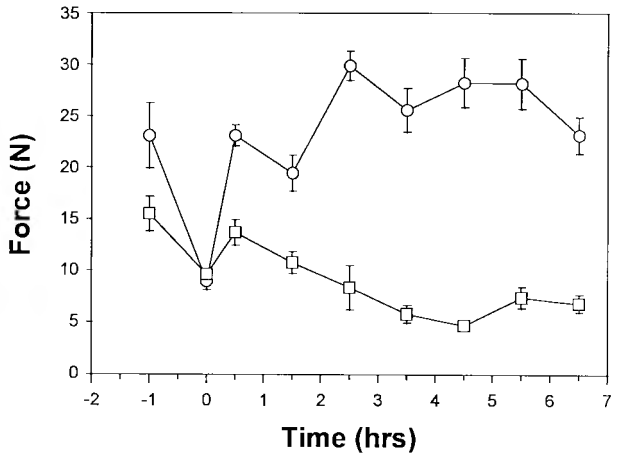

Fig. 3. *Decerebrate control.* Shown are tetanic forces generated by presenting 760 ms long 50 Hz trains of stimuli to two electrodes, separately, as a function of time. Anesthetic was discontinued at −2 h and decerebration was completed at time zero. Removal of the sodium pentobarbital anesthetic did not increase the excitability of the cord to electrical stimulation of ventral gray matter. Each point is the mean force from three to six tetanic contractions and error bars are the standard errors.

C. Decerebrate Preparation

Fig. 3 shows a record of pre- and postdecerebration forces from one cat. The two traces show tetanic forces generated by stimulating through two separate electrodes, respectively. The "0" mark on the horizontal axis refers to the time the decerebration was completed, over two hours after administration of Nembutal anesthetic had been discontinued. Each point is the mean of three to six tetanic contraction forces and the bars give the standard error. Immediately following decerebration the quadriceps force elicited from each site was depressed. The force returned to its predecerebration level within 30 min after decerebration and was maintained for six successive hours in one case (open circles), but seemed to degrade slightly in the other (open squares). For 6.5 h following the decerebration, stimulation of the spinal cord with 100 μA pulses caused no spread of activity to neighboring activation pools, as evidenced by the fact that no activity was recorded through the EMG electrodes or the foot pad transducer. Furthermore, stimulus trains presented to both electrodes in synchrony, while producing very large quadriceps forces (60.7 $\pm$ 3.3 N), caused no detectable spread of activity to motor neurons outside the quadriceps activation pool.

IV. Discussion

A. Electrode Interactions and Fatigue Reduction

A 1-mm spacing between electrodes resulted in minimal overlap of stimulated neuronal populations, even at the highest current level considered safe for intraspinal stimulation. On the other hand, this spacing provided for significant recruitment of additional motor neurons to produce large contraction forces with simultaneous stimulation through electrode pairs. These data correlate well with estimated values of effective current spread in the spinal cord gray matter, predicted to be in a range of 0.5 to 0.7 mm for 70 μA and 0.6 to 0.8 mm for 100 μA [19].

Muscle fatigue during electrical stimulation of motor nerves is due to several factors: reversed order of motor unit recruitment with the fatigue prone fibers activated first and for longer periods than fatigue resistant fibers; lack of topological selectivity of motor units within a muscle such that instead of rotating the mechanical work among several motor units, the stimulus continuously activates the same subset of fibers; and the use of synchronous stimulation of motor fibers with frequencies higher than physiological levels to produce large and fused muscular contractions [1], [3]–[5]. In an attempt to minimize these effects, investigators have developed novel stimulus waveforms to enhance near physiological size recruitment order of motor fibers [20], [21] and new electrode configurations to produce selective and topological recruitment of motor units [22]–[27].

We demonstrated elsewhere that focal spinal cord stimulation could recruit motor units in mixed order of size and presented data that suggested that one might be able to effect topological selectivity of motor units within a muscle by stimulating various portions of its activation pool [7], [28]. In the present study, we used dual channel stimulation to determine if stimulating independent sets of motor neurons, each at a rate below the fusion frequency, can decrease muscle fatigue while still producing fused tetani. Our results show that, though somewhat higher stimulus levels were required to produce initial force levels comparable to those produced by the single and refractory paradigms [17], [29], [30], fatigue was essentially eliminated by interleaved stimulation. As expected, the interleaved paradigm also produced greater post-tetanic potentiation due to the lower stimulation rate [31]–[34]. The ability to reduce fatigue by stimulating the spinal cord in an interleaved manner has the added benefit of activating the motor fibers in a manner that mimics their natural asynchronous firing pattern [1], [5]. This effect should be even stronger if more electrodes are used in a single muscle activation pool.

Evidence that the focal stimulation is activating motor neurons directly and is having little or no effect on interneurons in the cord comes from the decerebration experiment, in which the results of spinal cord stimulation remained largely the same under Nembutal anesthesia and for hours after anesthesia had been discontinued. Therefore, we believe that our observations can be reasonably extended to expected behavior in cases of lower limb paralysis in humans.

B. Proposed Specifications for a Spinal Cord Stimulation Electrode Array

Specifications for a stimulating electrode array for long term implantation in the human spinal cord can be developed based on the dimensions of the hamstrings, quadriceps, tibialis anterior and triceps surae activation pools, and the electrode interaction results presented in this paper. To do so, we have made a number of assumptions as follows. We assume that current spread in human ventral gray matter is similar to that in feline ventral horn. Given that the ventral horn motor pools in the human lumbo-sacral spinal cord possess the same spatial arrangement as those in the cat [14] and given the correspondence between activation and motor pools [7], we assume that the human lumbo-sacral spinal cord has the same arrangement of activation pools as does the cat. The lower extremity activation pools in humans are therefore primarily located in the lumbar enlargement between segments L2 and S2 [14]. The combined length of segments L2 to S2 in humans is approximately 50 mm and the width and height of the cord are approximately 15 and 12 mm measured at the midline and central canal level, respectively [35], [36]. This suggests an approximate 66% increase in the length and a 100% increase in the width and height of each activation pool, relative to the dimensions in cats.

TABLE I
SUMMARY OF ESTIMATED MOTOR ACTIVATION POOL LOCATIONS AND DIMENSIONS

Pool	Distance from Midline (mm)	Depth from Surface (mm)	Width (mm)	Height (mm)	Length (mm)
Hamstrings	3.6	7.6	1.0	1.5	19.0
Quadriceps	3.4	8.2	1.3	1.2	19.0
Tibialis Anterior	4.2	6.0	0.7	2.5	19.0
Triceps Surae	4.0	6.6	1.0	1.9	32.0

Ideally, an optimal electrode array would be one that would conform to the shape of each of the pools. However, the pools are irregular in shape and show variation between individuals. Therefore, as a matter of practicality, we have assumed a uniform cylindrical shape for each activation pool, with a width and height equal to that equaled or exceeded by at least 90% of the pool as observed in our detailed mapping experiments [7], [9]. These scaled dimensions are presented in Table I.

Assuming similar current spread characteristics in the cat and human spinal cord gray matter and symmetric current spread in all directions in the spinal cord ventral horn, and taking into consideration the sharing of lumbo-sacral segments between the pools, we propose a three-dimensional (3-D) array configuration to be implanted through the dorsal surface of the spinal cord. The rectangular array would consist of four rows of shafts, spaced 0.7 mm apart in the medial-lateral dimension. Each row, oriented along the rostro-caudal axis, would have 48 electrode shafts, spaced 1 mm apart. The shafts would be 8.8 mm long at the rostral end of the array, and 6.2 mm long at the caudal. The more rostral shafts would carry four 50 × 50 μm electrode sites spaced 1 mm apart, while the more caudal shafts would only need three sites. On each shaft, the most ventral electrode site would be 200 μm from the end of the shaft to allow for proper tapering of the electrode tip. The remaining sites would be spaced 1 mm apart relative to this ventral site.

The electrodes in such an array would provide access to fibers dispersed throughout each of the motor activation pools to ensure adequate rotation of stimulation among motor units in all muscle compartments. A 0.7-mm lateral spacing was chosen to coincide with the width of the tibialis anterior activation pool (the narrowest of the target pools) and to ensure the implantation of at least one comb in the pool. Implantation of electrodes close to the edges of the pool is not predicted to cause significant changes in the recruitment characteristics of the innervated muscle for two reasons. First, the pools

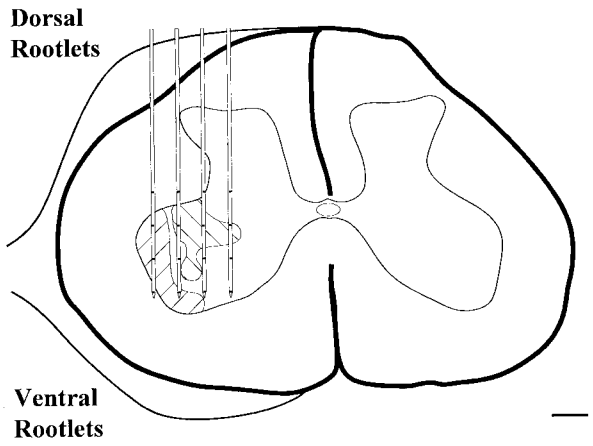

Fig. 4. *Sketch of cord cross section with proposed electrode array.* Shown are the experimentally determined tibialis anterior (ventro-lateral shaded area) and triceps surae (dorso-medial shaded area) activation pools at a single level of the spinal cord of a cat scaled up to assumed human proportions. Superimposed on the section is a scaled projection of where the shafts from the proposed electrode array would be with stimulation pads indicated by filled squares. The hamstring pool sits medial to the tibialis anterior pool and ventral to the triceps surae pool in the area where the ventro-medial electrode sites on the array are positioned. (Scale bar: 1 mm.)

were not mapped based on the morphological characteristics of their motor neurons but on their electrical characteristics [7]. With an imposed maximum current magnitude of 100 μA, no significant relationship was found in these studies between the location of a stimulated site in a pool, stimulation range (the extent to which stimulus strength can be increased relative to the threshold current before spread of activity to neighboring pools is detected), and generated muscle force. Second, Burke *et al.* indicated the presence of a sparse-cell layer dorsal and ventral to the medial gastrocnemius/soleus motor pool allowing for anatomical isolation of the pool [37]. It is expected that a similar situation exists for the remaining pools. Thus, with electrodes spaced 0.7 mm apart in a given pool, it is expected that motor neurons along the full width of the pool will be recruited with no spread to neighboring pools, even if the electrodes are not centered in the pool. Four rows of electrode combs and the lengths of the shafts were chosen to insure implantation of at least one comb in each of the target activation pools. The spacing of active sites on the electrode shafts was based on the electrode interaction data presented here, and the active electrode site dimensions were chosen to safely handle the charge injected (0.04 μC/phase at 1600 μC/cm^2) when maximal 100 μA, 400 μs duration stimuli are used [10].

Fig. 4 illustrates in cross section what the implanted array would look like in the lumbar spinal cord. The cord section and activation pools are taken from actual measurements in a single cat, and have been scaled up to human dimensions. Although not shown on this section because it was not mapped in this animal, the hamstrings pool would lie medial to the tibialis anterior pool and ventral to the triceps surae pool, so the electrode sites on the ventro-medial part of the array would lie in it.

With a shaft diameter of 100 μm the total tissue displacement by the array in the spinal cord would be less than 2%. The implanted array is expected to be subjected to little if any movement in paraplegic individuals for two reasons: 1) the lumbar enlargement in humans lies within thoracic vertebrae 9 to 12, an area which is stabilized by the rib cage and 2) movements about the hip such as flexion normally stretch the spinal roots instead of the lumbar spinal cord.

In addition to the benefits of stimulating motor neural tissue through multiple electrodes demonstrated in the present study as well as in studies by others [8], [12], [24], [25], [27], [38], a multi-electrode array of the type proposed here would provide a degree of fail-safe redundancy not possible with single electrode stimulation systems. Though the number of electrodes in the proposed array may appear large, it is still less than what is being proposed for visual prostheses electrode arrays. Since the latter is an active area of investigation in a number of labs, manufacturing techniques for the proposed array could be borrowed from those researchers, who are also actively engaged in developing multiplexing circuitry for accessing individual electrodes in such arrays [39]–[42].

At the expense of using more electrodes, stimulating the spinal cord for control of lower extremities has the added advantage of providing access to motor neurons innervating deep hip flexor and extensor muscles not easily accessible to cuff or motor point electrodes [5] and could be integrated with a spinal cord stimulation system for controlling bladder and bowel function [43].

ACKNOWLEDGMENT

The authors would like to thank A. Bird for her assistance in making the stimulating electrode arrays.

REFERENCES

[1] A. Prochazka, "Comparison of natural and artificial control of movement," *IEEE Trans. Rehab. Eng.*, vol. 1, pp. 7–16, 1993.
[2] P. A. Grandjean and J. T. Mortimer, "Recruitment properties of monopolar and bipolar epimysial electrodes," *Ann. Biomed. Eng.*, vol. 14, pp. 53–66, 1986.
[3] J. T. Mortimer, "Motor prostheses," in *Handbook of Physiology. Section 1: The Nervous System*, vol. II, Motor Control, Part 1, V. B. Brooks, Ed. Bethesda, MD: American Physiological Society, 1981, pp. 155–187.
[4] P. H. Peckham, J. P. Van Der Meulen, and J. B. Reswick, "Electrical activation of skeletal muscle by sequential stimulation," in *The Nervous System and Electrical Currents*, N. Wulfson and A. Sances, Jr., Eds. New York: Plenum, 1970, pp. 45–49.
[5] D. B. Popovic, "Functional electrical stimulation for lower extremities," in *Neural Prostheses: Replacing Motor Function After Disease or Disability*, R. B. Stein, P. H. Peckham, and D. B. Popovic, Eds. New York: Oxford Univ. Press, 1992, pp. 233–251.
[6] V. K. Mushahwar and K. W. Horch, "Selective activation of functional muscle groups through stimulation of spinal motor pools," in *Proc. 15th Annu. Int. Conf IEEE Eng. Med. Biol. Soc.*, San Diego, CA, 1993.
[7] V. K. Mushahwar, "Feasibility of spinal cord stimulation for control of lower extremities in paraplegia," Ph.D. dissertation, Univ. Utah, Salt Lake City, UT, 1996.
[8] K. Yoshida and K. Horch, "Selective stimulation of peripheral nerve fibers using dual intrafascicular electrodes," *IEEE Trans. Biomed. Eng.*, vol. 40, pp. 492–494, 1993.
[9] V. K. Mushahwar and K. W. Horch, "Selective activation of muscles in the feline hindlimb through electrical microstimulation of the ventral lumbo-sacral spinal cord," to be published.
[10] W. F. Agnew and D. B. McCreery, Neural Prostheses. Fundamental Studies. Englewood Cliffs, NJ: Prentice Hall, 1990.
[11] R. E. Burke, D. N. Levine, P. Tsairis, and F. E. Zajac, "Physiological types and histochemical profiles in motor units of the cat gastrocnemius," *J. Physiol.*, vol. 234, pp. 723–748, 1973.

[12] K. Yoshida and K. Horch, "Reduced fatigue in electrically stimulated muscle using dual channel intrafascicular electrodes with interleaved stimulation," *Ann. Biomed. Eng.*, vol. 21, pp. 709–714, 1993.
[13] G. J. Romanes, "The motor cell columns of the lumbo-sacral spinal cord of the cat," *J. Comparative Neurol.*, vol. 94, pp. 313–358, 1951.
[14] G. J. Romanes, "The motor pools of the spinal cord," *Progr. Brain Res.*, vol. 11, pp. 93–119, 1964.
[15] L. Bevan, Y. Loauris, R. M. Reinking, and D. G. Stuart, "The effect of the stimulation pattern on the fatigue of single motor units in adult cats," *J. Physiol.*, vol. 449, pp. 85–108, 1992.
[16] L. Bevan, Y. Laouris, S. J. Garland, R. M. Reinking, and D. G. Stuart, "Prolonged depression of force developed by single motor units after their intermittent activation in adult cats," *Brain Res. Bull.*, vol. 30, pp. 127–131, 1993.
[17] R. K. Powers and M. D. Binder, "Effects of low-frequency stimulation on the tension-frequency relations of fast-twitch motor units in the cat," *J. Neurophysiol.*, vol. 66, pp. 905–918, 1991.
[18] D. D. Hatcher and A. R. Luff, "Contractile properties of cat skeletal muscle after repetitive stimulation," *J. Appl. Physiol.*, vol. 64, pp. 502–510, 1988.
[19] E. Jankowska and W. J. Roberts, "An electrophysiological demonstration of the axonal projections of single spinal interneurons in the cat," *J. Physiol.*, vol. 222, pp. 597–622, 1972.
[20] Z.-P. Fang and J. T. Mortimer, "A method to effect physiological recruitment order in electrically activated muscle," *IEEE Trans. Biomed. Eng.*, vol. 38, pp. 175–179, 1991.
[21] R. Baratta, M. Ichie, S. K. Hwang, and M. Solomonow, "Orderly stimulation of skeletal muscle motor units with tripolar nerve cuff electrode," *IEEE Trans. Biomed. Eng.*, vol. 36, pp. 836–843, 1989.
[22] G. A. Baer, P. P. Talonen, V. Hakkinen, G. Exner, and H. Yrjola, "Phrenic nerve stimulation in tetraplegia," *Scand. J. Rehab. Med.*, vol. 22, pp. 107–111, 1990.
[23] G. A. Baer, P. P. Talonen, J. M. Shneerson, H. Markkula, G. Exner, and F. C. Wells, "Phrenic nerve stimulation for central ventilatory failure with bipolar and four-pole electrode systems," *PACE*, vol. 13, pp. 1061–1072, 1990.
[24] W. Happak, H. Gruber, J. Holle, W. Mayr, C. Schmutterer, U. Windberger, U. Losert, and H. Thoma, "Multi-channel indirect stimulation reduces muscle fatigue," in *Proc. Annu. Int. Conf., IEEE Eng. Med. Biol. Soc.*, Seattle, WA, 1989.
[25] H. Thoma, W. Girsch, J. Holle, and W. Mayr, "Technology and long-term application of an epineural electrode," *Trans. Amer. Soc. Artificial Internal Organs*, vol. 35, pp. 490–494, 1989.
[26] J. S. Petrofsky, "Sequential motor unit stimulation through peripheral motor nerves in the cat," *Med. Biological Eng. Comput.*, vol. 17, pp. 87–93, 1979.
[27] J. D. Sweeney, D. A. Ksienski, and J. T. Mortimer, "A nerve cuff technique for selective excitation of peripheral nerve trunk regions," *IEEE Trans. Biomed. Eng.*, vol. 37, pp. 706–715, 1990.
[28] V. K. Mushahwar and K. W. Horch, "Muscle recruitment through electrical stimulation of the lumbo-sacral spinal cord," to be published.
[29] M. Bergstrom and E. Hultman, "Contraction characteristics of the human quadriceps muscle during percutaneous electrical stimulation," *Pflugers Arch.*, vol. 417, pp. 136–141, 1990.
[30] D. Kernell, O. Eerbeek, and B. A. Verhey, "Relation between isometric force and stimulus rate in cat's hindlimb motor units of different twitch contraction time," *Experimental Brain Res.*, vol. 50, pp. 220–227, 1983.
[31] R. G. Cooper, R. H. T. Edwards, H. Gibson, and M. J. Stokes, "Human muscle fatigue: frequency dependence of excitation and force generation," *J. Physiol.*, vol. 397, pp. 585–599, 1988.
[32] H. Gibson, R. G. Cooper, M. J. Stokes, and H. T. Edwards, "Mechanisms resisting fatigue in isometrically contracting human skeletal muscle," *Quart. J. Experimental Physiol.*, vol. 73, pp. 903–914, 1988.
[33] H. J. Green and S. R. Jones, "Does post-tetanic potentiation compensate for low frequency fatigue?," *Clin. Physiol.*, vol. 9, pp. 499–514, 1989.
[34] S. A. Westwood, O. Hudlicka, and S. V. Perry, "Phosphorylation in vivo of the P light chain of myosin in rabbit fast and slow skeletal muscles," *Biochem. J.*, vol. 218, pp. 841–847, 1984.
[35] F. H. Netter, *The CIBA Collection of Medical Illustrations: Nervous System.* New York: CIBA, vol. 1, 1991.
[36] J. Nolte, *The Human Brain: An Introduction to its Functional Anatomy*, 3rd ed. St. Louis, MO: Mosby-Year Book, Inc., 1993.
[37] R. E. Burke, P. L. Strick, K. Kanda, C. C. Kim, and B. Walmsley, "Anatomy of medial gastrocnemius and soleus motor nuclei in cat spinal cord," *J. Neurophysiol.*, vol. 40, pp. 667–680, 1977.
[38] W. M. J. Grill and J. T. Mortimer, "Quantification of recruitment properties of multiple contact cuff electrodes," *IEEE Trans. Rehab. Eng.*, vol. 4, pp. 49–62, 1996.
[39] A. C. Hoogerwerf and K. D. Wise, "A three-dimensional microelectrode array for chronic neural recording," *IEEE Trans. Biomed. Eng.*, vol. 41, pp. 1136–1146, 1994.
[40] J. Ji, K. Najafi, and K. Wise, "A low-noise demultiplexing system for active multichannel microelectrode arrays," *IEEE Trans. Biomed. Eng.*, vol. 38, pp. 75–81, 1991.
[41] K. E. Jones, P. K. Campbell, and R. A. Normann, "A glass/silicon composite intracortical electrode array," *Ann. Biomed. Eng.*, vol. 20, pp. 423–437, 1992.
[42] C. T. Nordhausen, P. J. Rousche, and R. A. Normann, "Optimizing recording capabilities of the Utah intracortical electrode array," *Brain Res.*, vol. 637, pp. 27–36, 1994.
[43] W. F. Agnew, R. R. Carter, B. Woodford, D. B. McCreery, and L. A. Bullara, "Microstimulation of the lumbosacral spinal cord," Huntington Med. Res. Inst., Pasadena, CA, NIH NO1-NS-5-2333, Jan. 1–Mar. 31, 1996.

Vivian K. Mushahwar (S'92–M'97) received the B.S. degree in electrical engineering from Brigham Young University, Provo, UT, in 1991 and the Ph.D. degree in bioengineering from the University of Utah, Salt Lake City, UT, in 1996.

She is currently a Postdoctoral Fellow in the Department of Rehabilitation Medicine at Emory University, Atlanta, GA. Her research interests include identification of spinal systems responsible for locomotion, development of spinal cord neuroprostheses, and incorporation of motor control concepts in functional neuromuscular stimulation (FNS).

Dr. Mushahwar is a member of IEEE-EMBS, IFESS, and the Society for Neuroscience.

Kenneth W. Horch (M'88) received the B.S. degree from Lehigh University, Bethlehem, PA, and the Ph.D. from Yale University, New Haven, CT.

He is currently Professor of Bioengineering and of Physiology at the University of Utah, Salt Lake City. His research interests center on nerve repair and regeneration and neuroprosthetics.